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The Relationship between Gnawing and Food Consumption with Ventromedial Hypothalamic Lesions[1]

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COX, V. C., J. W. KAKOLEWSKI AND E. S. VALENSTEIN. *The relationship between gnawing and food consumption with ventromedial hypothalamic lesions.* PHYSIOL. BEHAV. 2 (4) 323–324, 1967.—Ventromedial hypothalamic lesions in rats increased food intake and decreased wood gnawing. Both feeding and gnawing can be elicited by electrical stimulation of the lateral hypothalamus. The opposite effect of ventromedial hypothalamic lesions on feeding and gnawing is discussed in terms of the relationship between the ventromedial hypothalamus and the lateral hypothalamus.

Ventromedial hypothalamic lesions Food intake Gnawing Ventromedial hypothalamus
Lateral hypothalamus

THE EFFECTS of ventromedial hypothalamic (VMH) damage on feeding behavior are well known. However, only a few studies have reported the effects of such neural damage on other behaviors. Kennedy and Mitra [4] reported decreased mating and general activity, and Grossman [2] and Sechzer, Turner and Liebelt [9] reported facilitation of avoidance conditioning following VMH damage. In the course of studying the effects of VMH damage on feeding we observed a decrease in wood gnawing. We pursued these observations systematically, because the hyperphagia following VMH damage is usually interpreted as a release of inhibition of the lateral hypothalamic "feeding area," but the effects of VMH damage on other behaviors elicited by stimulation of the lateral hypothalamus such as hoarding [3], coprophagia [5], paper shredding and wood gnawing [8] have received relatively little attention. The present study describes the results of an investigation of the effects of VMH damage on wood gnawing and food intake.

MATERIALS AND METHODS

Eight female albino rats (257–309 g) of the Holtzman strain were used. Prior to production of VMH lesions, daily baseline measures of food intake, water consumption, body weight, and wood gnawing were obtained. Powdered Purina Lab Chow was provided in a tunnel-device that permitted an accurate measurement of food consumption and spillage. The tunnel device was a Wahmann LC278 feeder modified by increasing the tunnel dia. to 3 in. Beechnut wood dowels 4 in. in length and 3/4 in. in dia. were used to measure wood gnawing. The dowels were weighed on a Mettler analytic balance at the time they were placed in the cage and 24 hr later. A fresh dowel was placed in each rat's cage daily. Animals were provided with dowels for 11 days prior to actual base-line measurements. After five days of base-line measurement all animals received bilateral electrolytic lesions of the VMH nucleus. The lesions were produced with the aid of a stereotaxic device and while the rats were anaesthetized with a mixture of chloral hydrate and pentobarbital. With the skull level between lambda and bregma the tip of the lesion probe was placed 2.50 mm posterior to bregma, 0.50 mm lateral to the midline and 0.50 mm from the bottom of the skull. The probe tip was bare of insulating enamel for 0.50 mm. The current used for the lesions was 2.0 mA d.c. delivered for 20 sec. After the lesions, measurement of food intake, water consumption, body weight, and gnawing was continued for 21 days.

RESULTS

Figure 1 presents the mean pre- and post-operative measures of body weight, food and water consumption, and gnawing for 5 pre- and the first 10 post-operative days. The same results were obtained for the remaining 11 days of measurement as well. Food and water consumption as well as body weight increased significantly after VMH lesions. In the first ten days following VMH damage, average daily food intake increased 68 per cent, average daily water consumption 114 per cent and body weight 18 per cent, when compared to pre-operative levels (For body weight, the formula $\frac{\text{Final Wt.} - \text{Projected Wt.}}{\text{Projected Wt.}} \times 100$

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was used. The projected weight was based on the average pre-operative weight gain.). In contrast, however, an 81 per cent decrease in gnawing after VMH lesions was observed. A Spearman rank-order correlation (r_s) between the percentage increase of post-operative food intake and percentage decrease in gnawing yielded a significant ($p < 0.05$) value of 0.78. These results indicate the electrolytic destruction of the VMH nucleus has an opposite effect on food intake and gnawing behavior. The decreased gnawing does not represent a general decline in vigor since data from our own laboratory and other studies [1, 10] have demonstrated increased VMH hyperphagia when the diet consisted of relatively hard Purina Lab Chow pellets.

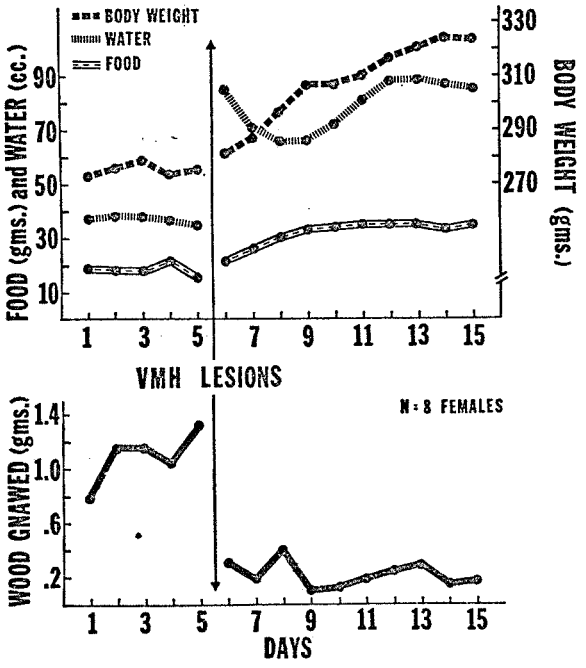

FIG. 1. Mean daily body weight, food and water consumption (top) and gnawing (bottom) prior to and after ventromedial hypothalamic lesions. Food available was powdered Purina Lab Chow.

DISCUSSION

Roberts and Carey [8] have demonstrated that stimulation of the lateral hypothalamus elicits wood gnawing if an appropriate stimulus is available. These authors demonstrated that wood gnawing could be elicited in the absence of feeding behavior. The wood gnawing sites are located in the same lateral hypothalamic area from which feeding can be elicited [6]. If the ventromedial hypothalamic area bears an inhibitory relationship to the lateral hypothalamus as some workers have suggested, then wood gnawing as well as food intake would be expected to increase following VMH damage. The present results indicate that if the VMH does inhibit the lateral hypothalamic area, it is restricted to specific neural systems. If the VMH exerts any direct effect on the lateral hypothalamic neural systems subserving wood gnawing it would seem to be of a facilitatory nature.

The results of the present study are also relevant to the "irritative" interpretation of VMH damage proposed by Reynolds [7]. The "irritative" hypothesis assumes that scar tissue formed by electrolytic lesions produces an irritative focus which stimulates adjacent viable tissue. Hyperphagia following VMH damage is assumed to be a function of stimulation of the adjacent lateral hypothalamic nucleus. If VMH damage activates adjacent lateral hypothalamic tissue such activation might be expected to increase wood gnawing as well as food intake as the lateral hypothalamic wood gnawing sites overlap with those producing feeding responses when stimulated [6, 8]. Consequently, unless one argues for a selective irritation process that activates lateral hypothalamic feeding, but inhibits gnawing sites, the present results are contrary to the "irritative" hypothesis.

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